

## Histopathological examination of a1 pulley tissue resected in trigger finger surgery and correlation with clinical results

Histopathology of a1 pulley tissue

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### Abstract

**Aim:** The aim was to analyze the histopathological features of the A1 pulley tenosynovial tissue surgically removed with the diagnosis of the trigger finger and to reveal its correlation with the clinic.

**Material and Methods:** Pathological tissue samples of 120 patients who underwent A1 pulley surgical resection with the diagnosis of trigger finger between January 2020 and December 2023 were retrospectively examined. The samples were evaluated with H&E, Alcian blue staining, and CD44 immunohistochemistry. The detected inflammation level, increased vascularization, degree of fibrosis, collagen structure, tenocyte proliferation, and hyalinization were scored semi-quantitatively. The relationship between histopathological findings and the clinic was evaluated statistically.

**Results:** In 81.7% of the examined cases, inflammation at different levels (mild 37.5%, moderate 30.0%, severe 14.2%) and increased vascularization were detected in 68.3%. In terms of fibrosis, varying degrees of involvement (mild 31.7%, moderate 40.0%, severe 18.3%) were observed in 90% of the cases. Collagen alignment was found to be disrupted in 71.7% of the cases. Tenocyte proliferation was positive in 76.7% and hyalinization was positive in 63.3%. A significant positive correlation was found between the degree of inflammation and VAS score ( $r = 0.684$ ,  $p < 0.001$ ) and between the degree of fibrosis and symptom duration ( $r = 0.712$ ,  $p < 0.001$ ).

**Discussion:** In the pathogenesis of the trigger finger, the presence of chondrocyte-like cells in the tenosynovial tissue, hypocellular collagen matrix containing hyaluronic acid, and inflammatory changes stand out as characteristic findings. A significant correlation was determined between the severity of these histopathological changes and clinical findings.

### Keywords

Trigger Finger, A1 Pulley, Tenosynovitis, Histopathology, Cd44, Hyaluronic Acid

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Introduction

Trigger finger (stenosing tenosynovitis) is a pathology that occurs with thickening and histomorphological changes in the fibrous structure of the flexor tendon sheath and A1 pulley. Accordingly, the movement functions of the flexor tendons are impaired, and clinical symptoms develop [1]. Histopathological studies have revealed complex tissue changes such as inflammatory changes, increased vascularization, and fibrosis in the A1 pulley tissue [2, 3].

When the A1 pulley is examined microscopically, its natural structure consists of regular collagen fibrils, minimal vascularity, and sparse fibroblasts. However, in the case of the trigger finger, disruption in the arrangement of collagen fibers, an increase in type III collagen, and significant proliferation in vascular structures are observed [4, 5]. Immunohistochemical studies have revealed infiltration of macrophages and increased expression of transforming growth factor-β (TGF-β) [6, 7].

In histopathological evaluations, acute inflammatory cell infiltration and vascular proliferation are evident in the early phase of the disease, while fibrosis and hyalinization are more prominent in the chronic phase. In particular, increased tenocytes and extracellular matrix changes play a key role in the worsening of the disease [8, 9, 10]. In analyses performed with semi-quantitative histopathological scoring systems, it was observed that these changes correlated with the clinical severity of the disease [11, 12].

Material and Methods

Patient Selection and Clinical Evaluation

In our study, 120 patients who underwent A1 pulley surgery due to a trigger finger in our clinic between January 2020 and December 2023 were retrospectively examined. Patients were staged preoperatively using Green’s classification [11]. Patients who had previously received corticosteroid injections due to trigger finger had inflammatory rheumatic diseases, and had undergone revision surgery on the same finger were excluded from our study [13].

Surgical Sample Collection and Tissue Follow-up

A1 pulley tissue samples we obtained through the surgical procedure were placed in 10% buffered formalin for pathological analysis. They were fixed for 24 hours. We performed tissue tracking as follows [8, 14]:

- Dehydration with alcohol series
- Clearing with xylol
- Paraffin application and blocking
- Taking 4 μm thick sections with a microtome
- H&E staining and Masson trichrome staining in selected preparations

Histopathological Examination

We evaluated the prepared sections according to the following criteria based on the clinical information of the patients [15, 16]:

- Inflammation Assessment:
  - oNone: Inflammatory cell infiltration
  - oMild: Focal minimal infiltration
  - oModerate: Widespread moderate infiltration
  - oSevere: Dense diffuse infiltration
- Other Parameters:

- oAssessment of increase in vascular structures
- oFibrosis degree (absent/mild/moderate/severe)
- oCollagen fiber pattern analysis
- oTenocyte proliferation
- oPresence of hyaline changes

In case of discrepancy, we made a mutual decision by evaluating with kappa analysis together with another pathologist at the microscope [17].

Statistical Analysis

The data obtained in this study were analyzed using SPSS Statistics 25.0 software. Descriptive statistics for continuous variables are presented as mean ± standard deviation, and for categorical variables as percentage (%).

To analyze the relationship between histopathological findings and clinical data:

- Spearman Correlation Analysis: To evaluate the nonlinear relationship between continuous variables (e.g., the relationship between the degree of inflammation and VAS score).
- Chi-Square Test: To analyze the relationship between categorical variables (e.g., the relationship between increased vascularization and the development of complications).
- Independent Sample T-Test: To compare the means of continuous variables between two groups (e.g., QuickDASH scores for groups with and without increased vascularization).
- Mann-Whitney U Test: To compare continuous variables that do not show normal distribution (e.g., the relationship between the degree of fibrosis and the duration of symptoms).
- Kappa Analysis: Used to measure the agreement between the evaluations between two pathologists.

The Kolmogorov-Smirnov Test was used to test normality distribution. Nonparametric tests were preferred for data that did not show normal distribution. The significance level in the analyses was accepted as p < 0.05.

Patient Selection

Our study was conducted retrospectively and 120 patients who underwent A1 pulley surgery due to trigger finger in our clinic between January 2020 and December 2023 were included in the study [11].

Inclusion Criteria:

- Being over 18 years of age
- Being diagnosed with trigger finger
- Accepting surgical treatment
- Committing to regular postoperative follow-ups

Exclusion Criteria:

- Having previously undergone trigger finger surgery on the same finger
- Having received local corticosteroid injection within the last 6 months
- Presence of rheumatoid arthritis or other inflammatory rheumatic diseases
- History of trauma/surgery on the wrist or finger
- Presence of carpal tunnel syndrome
- Presence of Dupuytren’s contracture
- Pregnancy
- Insufficient tissue sample for pathological examination [14, 15].

Ethical Approval

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Hospital (Date: 2025-01-02, No: 314).

Results

Demographic and Clinical Features

When the demographic data of 120 patients included in this study were examined, the mean age was found to be  $54.3 \pm 11.2$  years. More than two-thirds of the patients were women (68.3%, n = 82), while the proportion of men was 31.7% (n = 38). The mean time from the onset of symptoms to surgical intervention was calculated as  $8.4 \pm 4.2$  months, indicating a significant delay in surgical intervention. Dominant hand involvement was observed in 65% of the patients (n = 78), while non-dominant hand involvement was detected in 35% (n = 42). The mean body mass index (BMI) of the patients was  $27.8 \pm 4.6$  kg/m<sup>2</sup>, indicating that the patients were in the slightly obese class. The most common comorbid disease was Diabetes Mellitus, which was detected in 26.7% (n = 32) of the patients. This was followed by Hypertension (23.3%, n = 28) and Hypothyroidism (12.5%, n = 15). In the evaluation made according to occupational groups, it was determined that the majority of the patients were housewives (37.5%, n = 45), followed by office workers (26.7%, n = 32), workers (20.8%, n = 25) and retirees (15.0%, n = 18) (Table 1). The most commonly affected finger was the thumb (38.3%, n = 46), followed by the middle finger (26.7%, n = 32), index finger (23.3%, n = 28), ring finger (8.3%, n = 10) and little finger (3.3%, n = 4). The largest group consisted of Grade III patients (%43.3, n = 52). This was followed by Grade II (%31.7, n = 38), Grade IV (%15.0, n = 18) and Grade I (%10.0, n = 12) patients, respectively. In the preoperative functional evaluation, the mean QuickDASH score indicating the limitation in the patient's daily living activities was determined as  $48.6 \pm 12.4$ . In the pain evaluation, the mean VAS (Visual Analog Scale) score was  $7.2 \pm 1.8$ , indicating severe pain level. The mean hand grip strength was calculated as  $18.4 \pm 6.2$  kg and the range of motion of the affected finger was severely limited with an average of  $35.2 \pm 12.6$  degrees.

Table 1. Demographic and Clinical Characteristics (n=120)

	Number (n=120)	Percentage (%)
Age (years)	54.3 ± 11.2	-
Gender		
Female	82	68.3
Male	38	31.7
Affected Hand		
Dominant	78	65.0
Non dominant	42	35.0
BMI (kg/m²)	27.8 ± 4.6*	-
Comorbid Conditions		
Diabetes Mellitus	32	26.7
Hypertension	28	23.3
Hypothyroidism	15	12.5
Occupational Groups		
Homemaker	45	37.5
Office worker	32	26.7
Laborer	25	20.8
Retired	18	15.0

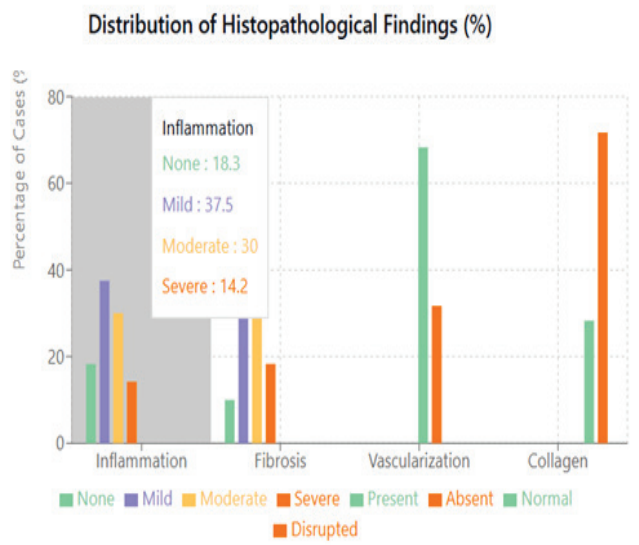


Figure 1. Distribution of Histopathological Findings (%)

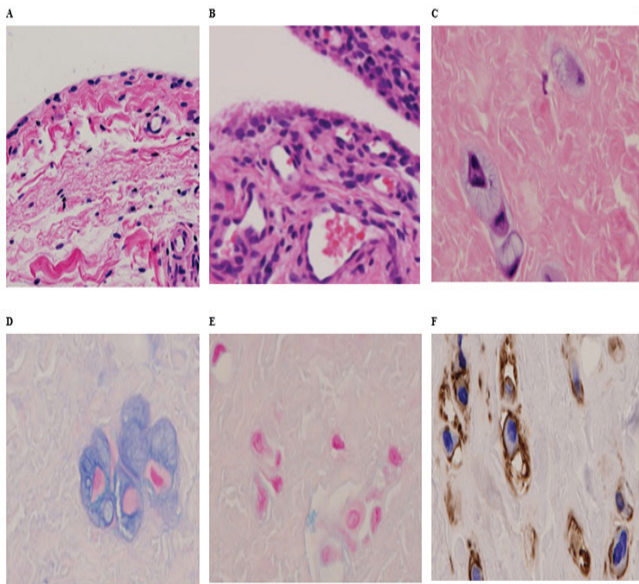


Figure 2. Histopathological and Immunohistochemical Analysis of A1 Pulley Tissue

Distribution of Fibrosis Grades According to Inflammation Severity

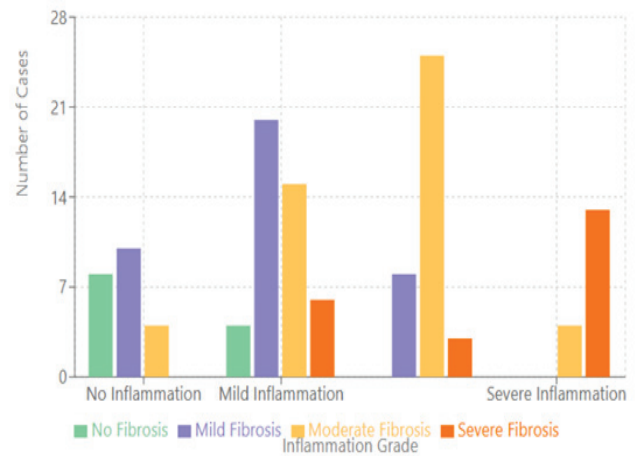


Figure 3. Correlation Between Inflammation Severity and Fibrosis Grades in Trigger Finger

### **Histopathological Examination Results**

In the microscopic evaluation of the resected A1 pulley tissue; In terms of inflammatory changes, varying degrees of inflammation were seen in the majority of cases (%81.7, n = 98). While the degree of inflammation was distributed as mild (%37.5, n = 45), moderate (%30.0, n = 36) and severe (%14.2, n = 17), inflammation was seen in only 18.3% (n = 22) of the cases. Proliferation in vascular structures was seen as positive in a significant majority of the patients (%68.3, n = 82). When the fibrotic structure was examined, varying degrees of fibrosis were detected in the majority of the cases (%90.0, n = 108). The distribution of fibrosis was as follows: moderate (%40.0, n=48), mild (%31.7, n = 38), and severe (%18.3, n = 22). Normal collagen formation was preserved in only 28.3% (n = 34) of the patients and was markedly irregular in the majority (71.7%, n = 86). Tenocyte proliferation was positive in more than three-quarters of the cases (76.7%, n = 92), and hyaline changes were positive in approximately two-thirds of the cases (63.3%, n = 76) (Figure 1).

In histopathological examination, a single-layer synovial cell layer was observed in the normal A1 pulley tissue (Figure 2A), while in trigger finger cases, significant inflammation and increased vascularization were observed (Figure 2B). Chondrocyte-like cells were also observed under the microscope (Figure 2C). Hyaluronic acid accumulation, which showed positive staining with Alcian blue, was observed around these cells (Figure 2D) and the staining disappeared after hyaluronidase application (Figure 2E). Chondrocyte-like cells showed positive expression with CD44 immunohistochemistry (Figure 2F).

### **Clinicopathological Correlation Analysis**

In our correlation analyses, a strong positive relationship was found between the degree of tissue inflammation and both pain scores ( $r = 0.684$ ,  $p < 0.001$ ) and functional disability level (QuickDASH score) ( $r = 0.625$ ,  $p < 0.001$ ). Fibrosis level showed a significant positive correlation with symptom duration ( $r = 0.712$ ,  $p < 0.001$ ), which revealed that fibrotic changes increased with the prolongation of disease duration. Pain scores were found to be significantly higher in patients with increased vascularization ( $r = 0.548$ ,  $p < 0.001$ ) (Figure 3).

### **Discussion**

Histopathological analysis of A1 pulley tenosynovial tissue is critical in understanding the pathogenesis of the trigger finger [1]. The microscopic findings and their clinical implications in our study provide very important data in terms of understanding the basis of the disease [17]. Recent literature studies also highlight the dynamic role of tenosynovial tissue [2].

### **Immunohistochemical Findings And Cellular Changes**

CD44-positive chondrocyte-like cells observed in tenosynovial tissue explain the cellular basis of pathogenesis [16]. S-100 protein negativity and CD44 positivity in these cells support the fibroblastic differentiation hypothesis in the literature [3]. These findings, which are parallel to the cell population defined by Yang et al. [6], exhibit explanatory features for the pathogenetic mechanism when considered together with the matrix organization study by Fritz et al. [7]. The tenosynovial tissue ultrastructure analysis by Ettema et al. [22] provides

biomechanical support for this mechanism.

### **Matrix Changes And Ultrastructural Analysis**

The hypocellular collagen matrix and hyaluronic acid accumulation we observed are quite similar to the study of Coronel et al. [10]. The standardized scoring of these changes was based on the criteria of Angrisani et al. [8], and it was found that there was a correlation with the ultrasonographic findings of Bianchi et al. [24].

### **Inflammatory Process And Vascularization**

Inflammatory patterns and vascular proliferation added new parameters to the scoring defined by Muthu et al. [17]. The chronic inflammation findings presented by Johnson et al. [18] are also parallel to the clinical progression analysis of Huang et al. [23]. These findings show significant differences from the normal tenosynovial structure described by Cohen et al. [4]. Our analysis of the relationship between time from symptom onset to surgery (mean  $8.4 \pm 4.2$  months) and inflammatory changes revealed that patients with longer waiting times tended to show more severe inflammatory patterns. Among patients with inflammation (81.7%), we observed a progression from mild (37.5%) to moderate (30.0%) and severe (14.2%) inflammation that corresponded with increasing duration of symptoms. This observation has important clinical implications, suggesting that prolonged waiting time for surgery may contribute to increased inflammatory severity. This finding aligns with Straszewski et al.'s [14] conclusions about the importance of surgical timing in managing inflammatory progression.

### **Clinical And Radiological Correlations**

The prognostic factors specified in the study of Belloti et al. [19] showed a strong correlation with our histopathological analysis results. These correlations confirm the hypotheses put forward in the early studies of Sbernadori et al. [5].

### **Treatment Implications**

The factors affecting treatment success highlighted in the meta-analysis of Lo et al. [20] can be explained by our findings obtained with histopathological analysis. The literature review of Markowitz et al. [21] emphasizes the role of histopathological changes in predicting treatment response. This approach is also in line with the evidence-based treatment recommendations of Amirfeyz et al. [11].

### **Methodological Approach And Standardization**

The standardized methodology we used adds strength to our study [8]. Our approach for analyzing advanced-stage cases aligns with recommended standards [25] and is consistent with the clinical evaluation criteria of Bridges et al. [12].

### **Future Perspectives**

The results obtained in our study support the conclusion that the clinical reflections of histopathological findings are decisive in the treatment of trigger fingers, emphasized in a recent review by Donati et al. [9]. The conservative treatment evaluation of Lunsford et al. [13] and the temporal relationship analysis between corticosteroid injection and surgical outcomes by Straszewski et al. [14] are guiding future research. It seems that the evaluation of postoperative complication rates in diabetic patients by Federer et al. [15] will contribute to the development of objective evaluation criteria. The histopathological patterns defined by Uchihashi et al. [2] may also shed light on new



treatment goals.

### Limitation

This study has the following limitations:

#### 1. Methodological Limitations:

- Retrospective study design
- A limited number of control tissue samples
- Inability to perform electron microscopic evaluation
- Collection of samples from a single center

#### 2. Technical Limitations:

- Analysis restricted to surgical specimens only
- Inability to study advanced molecular markers
- Limited immunohistochemical panel (only H&E, Alcian blue, and CD44)
- Absence of high-throughput molecular analyses
- Lack of fresh frozen tissue samples for specialized studies

#### 3. Clinical Limitations:

- Inability to evaluate preoperative radiological findings
- Absence of long-term follow-up data
- Unable to assess the effect of treatment response on histopathological changes
- Variation in the time interval between symptom onset and surgery

#### 4. Statistical Limitations:

- Small sample size in subgroup analyses
- Non-homogeneous distribution between diabetic and non-diabetic patient groups
- Limited power for correlation analyses in certain histopathological parameters

These limitations should be considered when interpreting the results, and future studies should address these constraints through more comprehensive and prospective evaluations with larger sample sizes and advanced molecular techniques.

### Conclusion

Our study provides important data in terms of a detailed description of histopathological changes in A1 pulley tissue in the pathogenesis of the trigger finger. Histopathological findings such as inflammation, fibrosis, increased vascularization and hyalinization in particular show a significant correlation with clinical symptoms. These results may contribute to the establishment of objective criteria in the staging of trigger finger disease.

In terms of clinical application, the findings obtained in our study can be evaluated as follows:

- The degree of inflammation and fibrosis may be enlightening in predicting the severity of a patient's clinical symptoms and their potential for recovery after surgery.
- Chondrocyte-like cells, especially detected by CD44 immunohistochemical staining, may provide opportunities for new treatment strategies targeting cellular targets.
- The effect of vascular proliferation on treatment results may suggest the need for revision of surgical techniques or additional complementary treatments.

Our recommendations for future research are as follows:

1. Investigation of Molecular Mechanisms: Studies investigating the genetic and biochemical basis of CD44-positive cells and extracellular matrix changes in understanding the pathogenesis of trigger finger disease may yield very good results.
2. Development of Treatment Approaches: The effect of these

histopathological findings on the response to treatment can be analyzed and patient-specific treatment approaches can be investigated.

3. Early Diagnostic Criteria: There is a strong relationship between clinical symptoms and histopathological findings. Early diagnostic criteria can be developed with advanced imaging and biomarker analyses.

In conclusion; this study draws attention to the importance of histopathological changes in trigger finger pathogenesis and makes valuable contributions to the clinical management process. New treatment strategies to be developed based on these findings in the future may improve the quality of life of patients and stop the progression of the disease.

### Scientific Responsibility Statement

The authors declare that they are responsible for the article's scientific content including study design, data collection, analysis and interpretation, writing, some of the main line, or all of the preparation and scientific review of the contents and approval of the final version of the article.

### Animal and Human Rights Statement

All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

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### Conflict of Interest

The authors declare that there is no conflict of interest.

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